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# New Subtype of Apical Hypertrophic Cardiomyopathy Identified With Nuclear Magnetic Resonance Imaging as an Underlying Cause of Markedly Inverted T Waves

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***Objectives.*** The aim of this study was to elucidate the clinical importance of a new subtype of apical hypertrophic cardiomyopathy that could not be diagnosed with the classical diagnostic criteria.

***Background.*** Apical hypertrophic cardiomyopathy is recognized by a characteristic spade-shaped intraventricular cavity on the end-diastolic left ventriculogram in the right anterior oblique projection, often associated with giant negative T waves [negativity ≥1.0 mV (10 mm)]. As an underlying cause of giant negative T waves, an additional new subtype of apical hypertrophic cardiomyopathy has been identified.

***Methods.*** In 40 patients with inverted T waves (negativity ≥0.5 mV), including 26 patients with giant negative T waves, nuclear magnetic resonance (NMR) long-axis images corresponding to the left ventriculogram in the right anterior oblique projection and short-axis images at various levels, including the apical level, were obtained to define the site of hypertrophied myocardium.

***Results.*** Long-axis images indicated a spadelike configuration in 17 patients, whereas this diagnostic configuration was not present in the other 23 patients. Nine of these 23 patients had significantly hypertrophied myocardium at the basal level. In the 14 remaining patients, short-axis images indicated no hypertrophy at the basal level and proved that the area of hypertrophied myocardium was confined to a narrow region of the septum or the anterior or lateral wall at the apical level (nonspade apical hypertrophic cardiomyopathy). The hypertrophied myocardium of the nonspade type was so narrowly confined that the mass did not form a spadelike configuration or could not be detected on the long-axis image.

***Conclusions.*** Nonspade apical hypertrophic cardiomyopathy was newly identified on NMR short-axis images, and this could be an additional, important underlying cause of moderately to severely inverted T waves.

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Giant negative T waves, defined as markedly inverted T waves with a negativity ≥1.0 mV (10 mm) (Fig. 1), were highlighted as a manifestation of apical hypertrophic cardiomyopathy in 1976 (1). This type of hypertrophic cardiomyopathy was shown to be characterized by presenting a spadelike configuration of the intraventricular cavity on the end-diastolic left ventriculogram in the right anterior oblique projection. The silhouette of the left ventricular cavity on this image resembles the spade in a deck of playing cards. Therefore, since 1979, the spadelike configuration has been the diagnostic criterion of apical hypertrophic cardiomyopathy (2), which has been recognized as part of the morphologically wide spectrum of hypertrophic cardiomyopathy (3–11). The spadelike configuration is defined on the left ventriculogram in the 30° right anterior oblique projection. Therefore, this configuration is the reflection of hypertrophied myocardium of the anterior wall and that of the posterior (inferior) wall at the apical level, with a lesser degree or no hypertrophy at the basal level of the left ventricle.

Recently, with the capability of nuclear magnetic resonance (NMR) imaging to provide accurate short-axis images at various levels, including the apical level, with clear margins of the endocardium and epicardium (12–14), various hypertrophy patterns at the apical level without significant hypertrophy at the basal level were demonstrated on short-axis images (14). There were two types of hypertrophy shown: One was confined to a small region of the septum or the anterior or lateral wall at the apical level (Fig. 2), and one was a diffusely circumferential hypertrophy at the apical level (Fig. 3). In the latter type, left ventriculography in the 30° right anterior oblique projection indicated a spadelike

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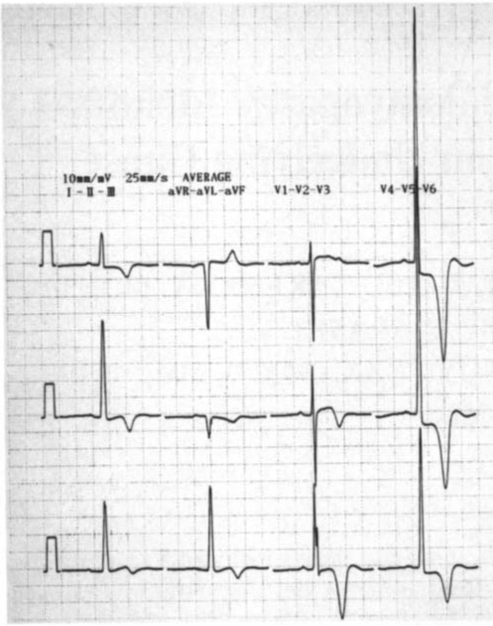
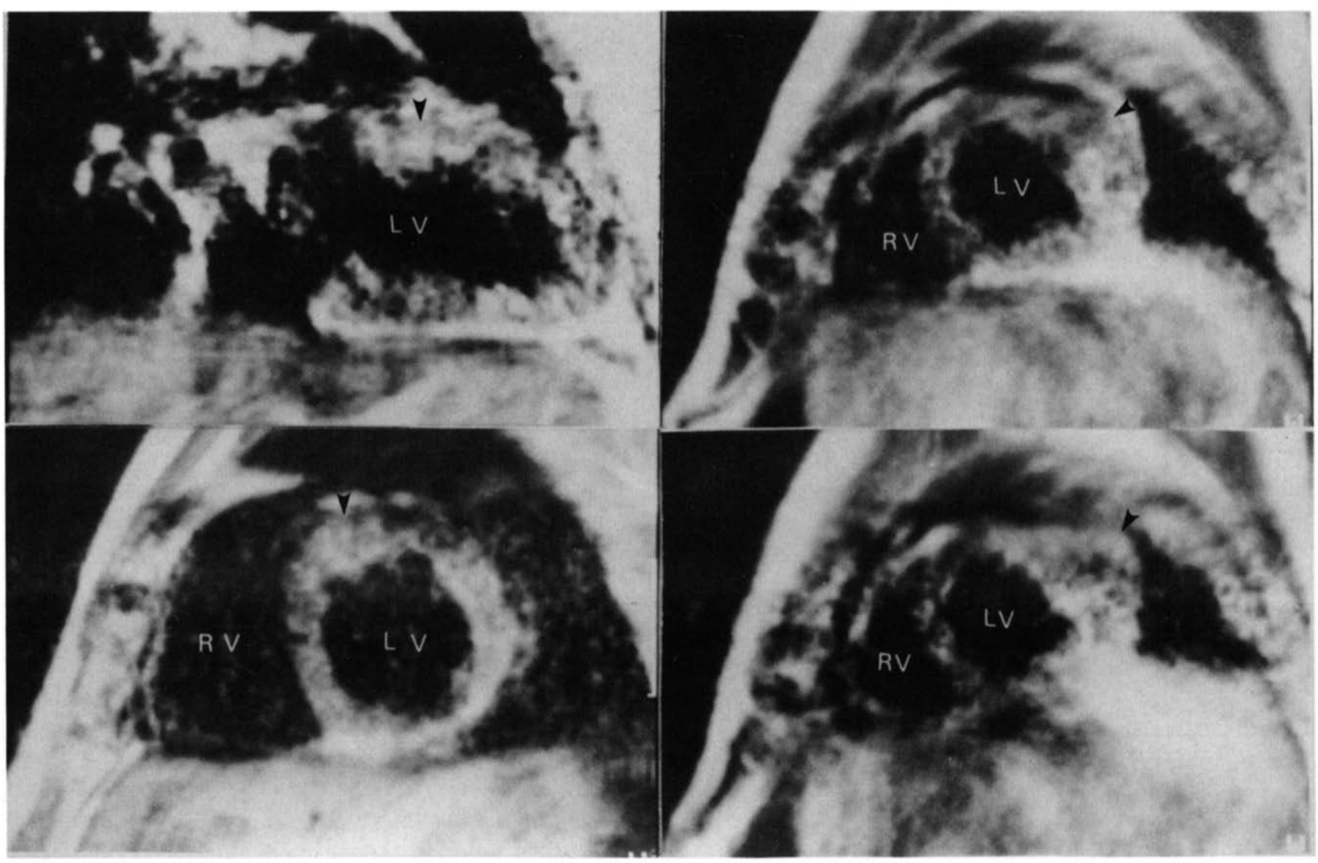



**Figure 1.** Electrocardiogram from a patient in Group A (T wave negativity ≥1.0 mV). Giant negative T waves with a high amplitude R wave of 7.4 mV (74 mm) are recorded in the precordial leads ($V_4$, $V_5$). The deepest T wave inversion is indicated in lead $V_4$, with a negativity of 2.8 mV.

configuration (referred to as spade apical hypertrophic cardiomyopathy herein). However, in the former type, left ventriculography did not indicate this diagnostic configuration or did not show any detectable hypertrophied myocardium in this projection (Fig. 4). On NMR short-axis images, we frequently encounter this new type of apical hypertrophic cardiomyopathy that cannot be diagnosed with the criterion for spade apical hypertrophic cardiomyopathy, not only in patients with giant negative T waves (Fig. 1) but also in patients with a lower negativity of T wave inversions (Fig. 5).

In the present study we tentatively termed this new type of apical hypertrophic cardiomyopathy the "nonspade" type (Fig. 2 and 4) and designed this study to elucidate the clinical importance of this morphologically new type as an additional

**Figure 2.** Nuclear magnetic resonance (NMR) images from a patient with nonspade apical hypertrophic cardiomyopathy. **Upper left,** Diastolic long-axis image corresponding to the left ventriculogram in the right anterior oblique projection. Although the quality of this NMR image is poor, it is clear that there is no significant hypertrophied myocardium at either the basal or the apical level. **Lower left,** End-diastolic short-axis image at the basal level. There is no significant hypertrophied myocardium at this level. **Upper right,** End-diastolic short-axis image at the apical level. The right and left intraventricular cavities with the lowest intensity, the low intensity narrow space of the intrapericardial cavity and the extracardiac fat with the highest intensity are clearly distinguished from the myocardium. **Lower right,** End-diastolic short-axis image for which the parallel imaging plane is nearer to the apical tip by 5 mm than that for the **upper right** short-axis image. Note that the localization of the hypertrophied myocardium is confined to the narrow part of the anterolateral wall at the apical level. **Arrows** = epicardium. LV = left ventricle; RV = right ventricle.



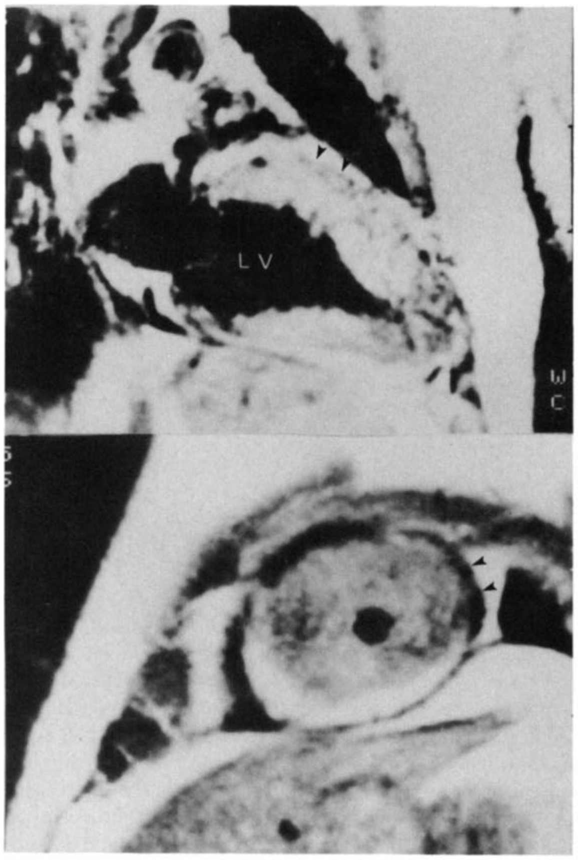



**Figure 3.** Nuclear magnetic resonace images from a patient with spade apical hypertrophic cardiomyopathy. **Upper panel,** Diastolic long-axis image corresponding to the left ventriculogram in the right anterior oblique projection that indicates a typical spadelike configuration. **Lower panel,** Short-axis image at the apical level that is obtained at end-diastole. Markedly and circumferentially hypertrophied myocardium is delineated. **Arrowheads** = pericardium. LV = left ventricle.

cause of moderately inverted T waves as well as giant negative T waves on the electrocardiogram (ECG).

## Methods

**Study patients.** The study group consisted of 40 patients with T wave inversions and a high QRS voltage ($RV_5 \geq 2.7$ mV or $SV_1 + RV_5 \geq 3.5$ mV) in the precordial leads of the ECG who were thought to have hypertrophic cardiomyopathy on the basis of these ECG findings and clinical history. They gave informed consent for this study (Table 1). The negativity of T wave inversions in the left precordial leads was ≥0.5 mV (5 mm) in all 40 patients. The subjects were assigned to two groups according to the negativity of the T wave inversions. Twenty-six patients had giant negative T waves (1,2) with a negativity ≥1.0 mV (Group A, Fig. 1), and the negativity of T waves in the other 14 patients was ≥0.5 and <1.0 mV (Group B, Fig. 5). No patient had a history of symptoms or signs suggesting ischemic heart disease, including myocardial infarction (coronary angiography in seven patients in Group A showed normal coronary arteries) (15) or a cerebrovascular accident (16). No patient had a condition that might cause left ventricular hypertro-

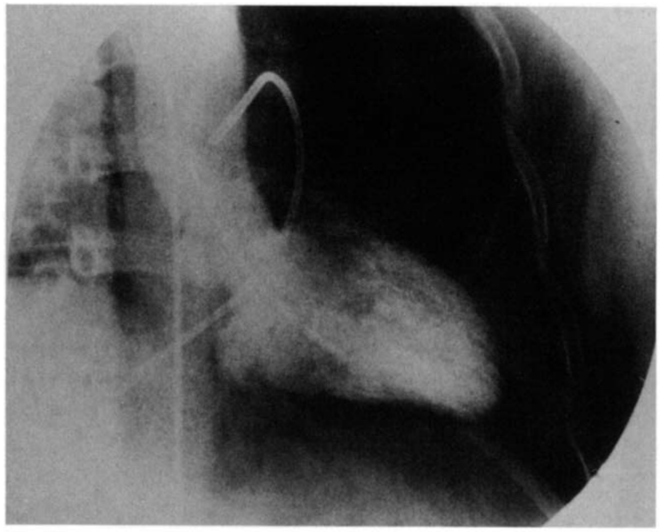


**Figure 4.** Thirty-degree right anterior oblique left ventriculogram at end-diastole in a patient in Group A with nonspade apical hypertrophic cardiomyopathy. Although this patient has hypertrophied muscle at the small region of the anterolateral wall at the apical level, the ventriculogram shows no hypertrophied myocardium detectable in this plane.

**Figure 5.** Electrocardiogram from a patient in Group B (T wave negativity ≥0.5 mV and <1.0 mV). Moderately inverted T waves are shown in leads $V_4$, $V_5$ and $V_6$ (negativity 0.8 mV).

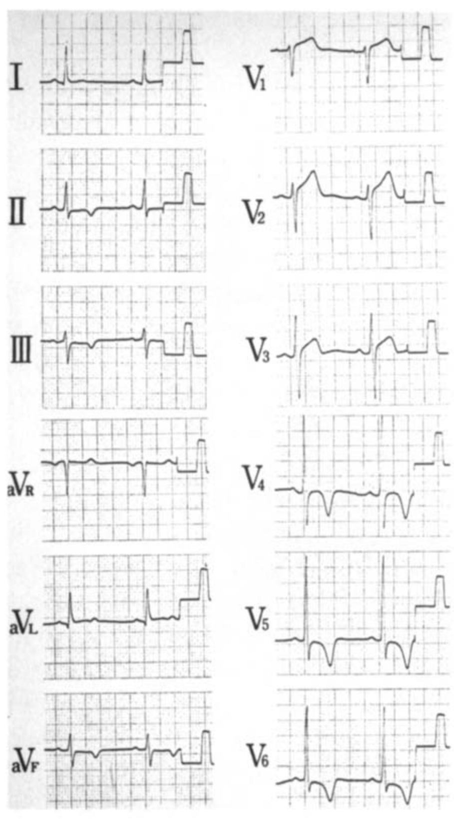


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Table 1. Study Subjects

| | Group A (n = 26) | Group B (n = 14) |
|---|---|---|
| Age (yr) | 58.8 ± 10.6* | 50.2 ± 9.4 |
| Negativity of T wave (mV) | 1.48 ± 0.52 | 0.74 ± 0.14 |
| Amplitude of R wave (mV) | 3.92 ± 1.36† | 2.70 ± 0.50 |

*$p < 0.05$. †$p < 0.01$. Values presented are mean value ± SD. Group A = T wave negativity ≥1.0 mV; Group B = T wave negativity ≥0.5 and <1.0 mV.

phy, including valvular heart disease. The mean age, mean values of the negative T waves and R wave voltage in the common precordial lead in Group A and B patients are noted in Table 1.

**Nuclear magnetic resonance imaging.** All NMR images were obtained with a 1.5-T superconducting magnet (MAGNETOM, Siemens). The imaging sequence used was an ECG-gated spin-echo with a flip angle of 90° and an echo time of 34 ms. The minimal time delay for the 90° pulse of selective excitation after the triggering of the R wave on the ECG was 40 ms with this machine. Therefore, 74 ms after the trigger point was the time point at which the first echo was recorded as an end-diastolic image (17). The imaging planes were the left ventricular long-axis plane, which corresponded to the left ventriculogram in the 30° right anterior oblique projection, and the left ventricular short-axis planes at the basal and apical levels. The basal level was set at the level of the chordae tendineae of the mitral valve and the distance between the imaging plane for the apical short-axis image, and the tip of the apex of the left ventricle was 2.5 cm. The slice thickness was 10 mm, and the reconstruction matrix was 256 × 256. Details of the imaging methods used in the present study have been described elsewhere (14,17).

**Measurements.** On NMR end-diastolic short-axis images at the basal and apical levels, the myocardial wall of the left ventricle was divided into four segments: anterior, lateral, posterior and septal segments at the basal and apical levels (Fig. 6). All measurements were performed on these short-axis images obtained at end-diastole.

**Definition of spade and nonspade apical hypertrophic cardiomyopathy.** For the apical level wall, a thickness ≥15 mm was considered to indicate hypertrophy. The wall thickness at the apical level in normal subjects was 8.9 ± 1.9 mm in our previous study (14). Therefore, 15 mm (mean + 3 × SD = 8.9 mm + 3 × 1.9 mm = 14.6 mm < 15 mm) was used as the cutoff for hypertrophy at the apical level.

Spade-type apical hypertrophic cardiomyopathy was defined as follows: One or more segments at the apical level were recognized as "hypertrophied," and the ratio (Ra) of the anterior wall thickness at the apical level to the anterior wall thickness at the basal level was ≥1.3, according to the original 1979 definition (2) of spadelike configuration on left ventriculography (Fig. 6). Moreover, the silhouette of the intraventricular cavity of the left ventricle on end-diastolic NMR long-axis images that corresponded to the left ventriculogram in the right anterior oblique projection was judged to be a spadelike configuration on visual inspection by two independent observers (Fig. 3).

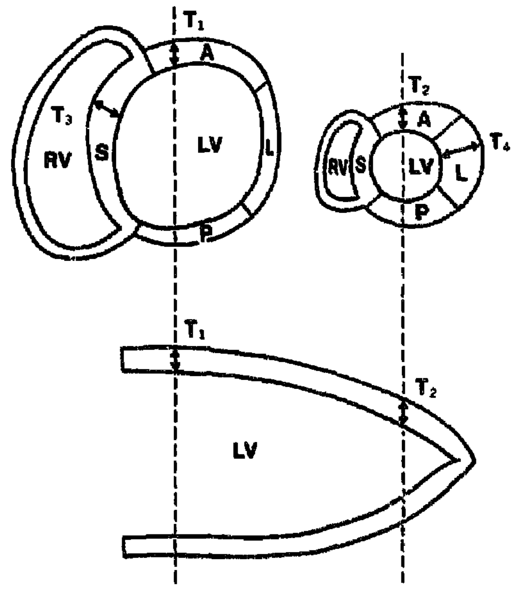


**Figure 6.** Measurements and definition of ratios used for diagnostic criteria. **Upper left,** Scheme of the short-axis image at the basal level. **Upper right,** Scheme of the short-axis image at the apical level. **Lower scheme,** Long-axis image that corresponds to the left ventriculogram in the right anterior oblique projection. The imaging planes for the nuclear magnetic resonance (NMR) short-axis images for the apical and basal levels are indicated on the **lower scheme** by the **dotted lines.** The basal level was set at that of the mitral chordae tendineae, and the distance between the imaging plane for the apical short-axis image and the tip of the apex was 2.5 cm. The imaging plane for the NMR long-axis image corresponding to the left ventriculogram in the right anterior oblique projection is indicated by the **dotted lines** on the two short-axis images. Note that only the anterior and posterior walls can be delineated on the long-axis image. These schemes represent the hypertrophied myocardium that was confined to the lateral wall at the apical level in a patient with nonspade apical hypertrophic cardiomyopathy. On the long-axis image, no hypertrophied myocardium is detected. The ratio (Ra) of the anterior wall thickness at the apical level to the anterior wall thickness at the basal level, which is used for the diagnosis of spade apical hypertrophic cardiomyopathy, is defined as $Ra = T_2/T_1$. The ratio (Rm) of the maximal wall thickness at the apical level to the maximal wall thickness at the basal level, which is the diagnostic criterion for nonspade apical hypertrophic cardiomyopathy, is defined as $Rm = T_4/T_3$. A = anterior wall; L = lateral wall; LV = left ventricle; P = posterior wall; RV = right ventricle; S = septum; $T_1$ = wall thickness of the anterior wall at the basal level, which can be evaluated on the short- and long-axis images; $T_2$ = wall thickness of the anterior wall at the apical level, which can be assessed on the short- and long-axis images; $T_3$ = maximal wall thickness at the basal level; $T_4$ = maximal wall thickness at the apical level.

The maximal wall thicknesses at the apical and basal levels were determined on NMR short-axis images obtained at the apical level and the basal level, respectively (Fig. 6). Nonspade apical hypertrophic cardiomyopathy was tentatively defined by the following criteria in the present study.

**Table 2.** Wall Thickness in Group A (T wave negativity ≥1.0 mV)

| | Spade (n = 15) | Nonspade (n = 8) | Other (n = 3) |
|---|---|---|---|
| Basal (mm) | | | |
| Sep | 10.9 ± 1.8 | 11.6 ± 1.5 | 13.0 ± 1.0 |
| Ant | 13.4 ± 1.6 | 11.8 ± 1.0 | 15.3 ± 2.1 |
| Lat | 11.0 ± 1.7 | 10.9 ± 1.6 | 11.3 ± 2.1 |
| Pos | 9.9 ± 2.1 | 10.1 ± 2.0 | 10.0 ± 1.0 |
| Apical (mm) | | | |
| Sep | 17.3 ± 3.3 | 12.6 ± 3.0 | 14.0 ± 2.6 |
| Ant | 20.5 ± 2.7 | 15.5 ± 5.6 | 15.3 ± 1.5 |
| Lat | 18.9 ± 3.4 | 15.5 ± 3.8 | 14.7 ± 2.5 |
| Pos | 12.7 ± 2.5 | 9.5 ± 2.5 | 10.0 ± 0.0 |

Values presented are mean value ± SD. Ant = anterior wall; Apical = apical level; Basal = basal level; Lat = lateral wall; Nonspade = nonspade apical hypertrophic cardiomyopathy; Other = hypertrophic cardiomyopathy other than apical types, including hypertrophic obstructive and nonobstructive cardiomyopathies; Pos = posterior wall; Sep = septum; Spade = spade apical hypertrophic cardiomyopathy.

The maximal wall thickness at the apical level was ≥15 mm, and the ratio (Rm) of the maximal wall thickness at the apical level to the maximal wall thickness at the basal level was ≥1.3 (Fig. 6). Moreover, the NMR long-axis images corresponding to left ventriculogram in the right anterior oblique projection either did not show any hypertrophied myocardium or indicated a small amplitude of hypertrophied myocardium that was not massive enough to form a spadelike configuration. This ratio Rm was different from that of Ra used as the criterion for spade apical hypertrophic cardiomyopathy because the anterior segment at the apical level of nonspade apical hypertrophic cardiomyopathy was not necessarily hypertrophied (Fig. 2, 4 and 6).

**Statistical analysis.** Values were expressed as mean value ± 1 SD. The unpaired *t* test was used to test differences between the two groups. A significant difference was indicated by $p < 0.05$.

**Table 3.** Wall Thickness in Group B (T wave negativity ≥0.5 and <1.0 mV)

| | Spade (n = 2) | Nonspade (n = 6) | Other (n = 6) |
|---|---|---|---|
| Basal (mm) | | | |
| Sep | 12.5 ± 2.1 | 10.8 ± 1.8 | 13.7 ± 2.1 |
| Ant | 16.5 ± 0.7 | 12.5 ± 1.6 | 19.2 ± 5.5 |
| Lat | 13.0 ± 1.4 | 12.5 ± 1.4 | 15.0 ± 4.6 |
| Pos | 10.5 ± 2.1 | 9.0 ± 1.7 | 11.0 ± 0.9 |
| Apical (mm) | | | |
| Sep | 11.0 ± 1.4 | 9.8 ± 2.9 | 17.2 ± 6.1 |
| Ant | 22.5 ± 2.1 | 13.7 ± 3.1 | 17.2 ± 2.4 |
| Lat | 23.0 ± 1.4 | 19.5 ± 4.6 | 13.5 ± 3.7 |
| Pos | 12.0 ± 0.0 | 9.5 ± 1.9 | 11.0 ± 2.5 |

Values presented are mean value ± SD. Abbreviations as in Table 2.

**Table 4.** Origin of Negative T Waves

| | Group A (n = 26) (negativity ≥1.0 mV) | Group B (n = 14) (0.5 ≤ negativity < 1.0) |
|---|---|---|
| Spade | 15 | 2 |
| Nonspade | 8 | 6 |
| Others | 3 | 6 |

Abbreviations as in Table 2.

## Results

The end-diastolic wall thickness of each segment is shown in Tables 2 and 3.

In Group A (negativity of T wave inversion ≥1.0 mV), 15 patients were diagnosed as having spade apical hypertrophic cardiomyopathy, whereas 8 were judged to have nonspade apical hypertrophic cardiomyopathy. Although the three remaining patients in this group had hypertrophied segments at the apical level, they did not meet the criteria for spade or nonspade apical hypertrophic cardiomyopathy (Table 4). These three patients had significant hypertrophy at the basal level that was comparable to or greater than that at the apical level. Therefore neither the ratio (Ra) of wall thickness of the apical anterior segment to that of the basal anterior segment nor the ratio (Rm) of the maximal wall thickness at the apical level to that at the basal level exceeded 1.3 (Table 5).

In all 15 patients who were diagnosed with spade apical hypertrophic cardiomyopathy in Group A, the ratio Rm was ≥1.3 (Table 5), and the segment at the apical level with the greatest degree of hypertrophy was the septal segment in 2 patients, the anterior segment in 7 and the lateral segment in 6.

In three of the eight patients who were diagnosed with nonspade apical hypertrophic cardiomyopathy in Group A, the ratio Ra was >1.3 (Table 5). However, because of the weak amplitude of hypertrophy of the posterior (inferior) segment at the apical level, the NMR long-axis images corresponding to the left ventriculogram in the right anterior oblique projection did not indicate a spadelike configuration. The segment that showed the maximal amplitude of hypertrophy at the apical level was the septal segment in two

**Table 5.** Ratio of Diagnostic Criterion

| | Group A | | | Group B | | |
|---|---|---|---|---|---|---|
| | Spade (n = 15) | Nonspade (n = 8) | Other (n = 3) | Spade (n = 2) | Nonspade (n = 6) | Other (n = 6) |
| Rm ≥1.3 | 15 | 8 | 0 | 2 | 6 | 0 |
| Rm <1.3 | 0 | 0 | 3 | 0 | 0 | 6 |
| Ra ≥1.3 | 15 | 3 | 0 | 2 | 1 | 0 |
| Ra <1.3 | 0 | 5 | 3 | 0 | 5 | 6 |

Ra = ratio of the wall thickness of the anterior wall at the apical level to that at the basal level; Rm = ratio of the maximal wall thickness at the apical level to that at the basal level. Other definitions and abbreviations as in Tables 1 and 2.

patients, the anterior segment in three and the lateral segment in three.

In Group B (negativity of T wave inversion ≥0.5 and <1.0 mV), two patients were diagnosed with spade and six with nonspade apical hypertrophic cardiomyopathy. The amplitude of hypertrophy at the basal level of the six remaining patients in this group was equal to or greater than that at the apical level. Therefore, these six patients were classified as having hypertrophic cardiomyopathy other than the two apical types (Table 4).

The ratio Rm was ≥1.3 in the two patients that showed a spadelike configuration in Group B (Table 5). The greatest hypertrophied segment at the apical level was the anterior segment in one patient and the lateral segment in the other.

In five of the six patients with nonspade apical hypertrophic cardiomyopathy in Group B, the ratio Ra was <1.3 because of the small amplitude of hypertrophy of the anterior wall at the apical level (Table 5). The segment that represented the maximal wall thickness at the apical level was the anterior segment in one patient and the lateral segment in the other five patients. Both ratios of Ra and Rm were <1.3 in the six patients who were diagnosed with hypertrophic cardiomyopathy other than the apical types in the Group B (Table 5).

## Discussion

**Nonspade apical hypertrophic cardiomyopathy.** This is the first description of the morphology of a new type of hypertrophic cardiomyopathy tentatively termed nonspade apical hypertrophic cardiomyopathy. In the present study, we demonstrated that the nonspade type might also develop giant negative T waves with a negativity ≥1.0 mV, occasionally up to 1.5 mV, and that the nonspade type could be one of the most important underlying disorders for negative T waves, especially in patients with moderately inverted T waves (0.5 mV ≤ negativity < 1.0 mV). We also call attention to the pitfall that this new type of apical hypertrophic cardiomyopathy cannot be diagnosed by the classical diagnostic criterion used for spade apical hypertrophic cardiomyopathy or any hypertrophied myocardium cannot be detected on the long-axis images corresponding to the left ventriculogram in the 30° right anterior oblique projection. In hypertrophic cardiomyopathy with asymmetric septal hypertrophy, predominantly hypertrophied myocardium is usually located at the basal level. Accordingly, the ratio (Rm) of the maximal wall thickness at the apical level to that at the basal level might be <1.0. Moreover, in our previous study, Rm in normal subjects was <1.0. Therefore, the arbitrary criterion of Rm ≥1.3 was tentatively used for the diagnosis of nonspade apical hypertrophic cardiomyopathy in the current study.

**Characteristic electrocardiographic findings in apical hypertrophic cardiomyopathy.** Acute myocardial infarction (15) and cerebrovascular accidents (16) might induce giant negative T waves in the left precordial leads on ECG. In myocardial infarction, the amplitudes of the R waves are decreased in the same leads that indicate markedly inverted T waves. Conversely, characteristic ECG findings in patients with apical hypertrophic cardiomyopathy were giant negative T waves associated with high QRS voltage and lesser degree of ST segment depression (Fig. 1).

**Diversity of the hypertrophy pattern in apical hypertrophic cardiomyopathy.** With the use of NMR short-axis images, we previously demonstrated diversity of the localization of hypertrophied myocardium at the apical level in patients with apical hypertrophic cardiomyopathy who did not have significant hypertrophy at the basal level or did not have basal hypertrophy comparable to that at the apical level (14). The localization of the hypertrophied myocardium was not necessarily circumferential at the apical level and was often confined to the small region of the septum or the anterior or lateral wall at the apical level.

**Nonspade apical hypertrophic cardiomyopathy cannot be diagnosed with the classical criteria.** The anatomic characteristic of classical apical hypertrophic cardiomyopathy has been the spadelike configuration of the intraventricular cavity on the end-diastolic left ventriculogram in the right anterior oblique projection. Therefore, this configuration was used as diagnostic criterion for apical hypertrophic cardiomyopathy (2). On the left ventriculogram in the right anterior oblique projection, the anterior and the posterior (inferior) walls of the left ventricle can be visualized, whereas, neither the interventricular septum nor the lateral wall can be delineated in this projection. Therefore, if the hypertrophied myocardium is confined to the small region of the septum or the lateral wall at the apical level, it might not be possible to detect any hypertrophied myocardium on this image (Fig. 2, 4, and 6). Left ventriculograms in the right anterior oblique projection or NMR long-axis images corresponding to the left ventriculograms in this projection do not indicate a spadelike configuration in some patients with apical hypertrophic cardiomyopathy because hypertrophied myocardium is confined to small regions that cannot be delineated on the long-axis images.

**Nuclear magnetic resonance imaging to evaluate hypertrophy at the apex.** The discovery of nonspade apical hypertrophic cardiomyopathy was largely due to the characteristic capability of NMR imaging to circumferentially delineate the apex. With two-dimensional echocardiography it might be difficult to obtain accurate short-axis images of the apex, and it might be also difficult to delineate clear margins of both the endocardium and the epicardium at the apical level of the left ventricle. With the use of left ventriculography, even with biplane methods, circumferential assessment of the left ventricular wall thickness at the apex might not be possible. Nuclear magnetic resonance imaging can provide clear margins of both the endocardium and the epicardium of the apex, and with this imaging technique the exact short-axis planes can be obtained at various levels, including the apical level (12–14,17–19).

**Clinical importance of nonspade apical hypertrophic cardiomyopathy.** In the present study, we assigned the subjects to two study groups according to the severity of negative T waves. In patients with giant negative T waves, 30% were diagnosed with nonspade apical hypertrophic cardiomyopathy. Moreover, in >40% of patients with moderately inverted T waves with a negativity ≥0.5 and <1.0 mV, the underlying disorder was nonspade apical hypertrophic cardiomyopathy. Because the study patients were not selected in a strictly consecutive manner, we could not obtain in our study a determination of the prevalence of each type of cardiomyopathy that might be directly applicable to the general population. However, it is possible that nonspade apical hypertrophic cardiomyopathy constitutes an important cause of the moderately to severely inverted T waves in the precordial leads. Spade and nonspade apical hypertrophic cardiomyopathies are clinically significant in that both could be an important underlying cause of markedly inverted T waves. Whether both types of apical hypertrophic cardiomyopathy might be included in the common disease entity in the common spectrum of hypertrophic cardiomyopathy remains to be resolved in the future. However, at present it should be stressed that it is clinically important to separate apical hypertrophic cardiomyopathy into the two entities (i.e., spade and nonspade) because the latter cannot be diagnosed with the classical criteria of the spadelike configuration, and any hypertrophied myocardium of the latter type cannot be detected on long-axis images without scrutinizing the apex of the left ventricle circumferentially.

The sensitivity and specificity of the diagnosis of spade or nonspade apical hypertrophic cardiomyopathy with echocardiography were not estimated in the current study. Lack of this information may be a weakness of the current study because echocardiography is the standard screening test in most centers.

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